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(FILE 'HOME' ENTERED AT 14:08:22 ON 03 MAR 2004)

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
     14:08:42 ON 03 MAR 2004
L1             0 S HEMATOPOIESIS AND (BASOPHIL ACTIVAT?)
L2         74090 S HEMATOPOIESIS
L3           639 S (BASOPHIL ACTIVATION)
L4             0 S L2 AND L3
L5           579 S L2 AND BASOPHIL?
L6           110 S L5 AND MEGAKARYOC?
L7            29 S L6 AND MAST?
L8            29 S L7 AND HEMATO?
L9            13 DUPLICATE REMOVE L8 (16 DUPLICATES REMOVED)
L10            2 S L9 AND ACTIVA?

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
     14:22:10 ON 03 MAR 2004
L11      1074228 S HEMATO?
L12            8 S L9 AND STIMU?
L13            6 S L12 NOT L10

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(FILE 'HOME' ENTERED AT 14:08:22 ON 03 MAR 2004)

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L11      1074228 S HEMATO?
L12            8 S L9 AND STIMU?
L13            6 S L12 NOT L10
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ANSWER 4 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1994:215037 CAPLUS
DN 120:215037
ED Entered STN: 30 Apr 1994
TI Lymphoid and myeloid differentiation of single human CD34+, HLA-DR+, CD38- **hematopoietic** stem cells
AU Huang, Shiang; Terstappen, Leon W. M. M.
CS Becton Dickinson Immunocytometry Syst., San Jose, CA, 95131, USA
SO Blood (1994), 83(6), 1515-26
CODEN: BLOOAW; ISSN: 0006-4971
DT Journal
LA English
CC 15-5 (Immunochemistry)
Section cross-reference(s): 2, 13
AB Multilineage differentiation of human fetal bone marrow CD34+ cell subsets was examined using a single-cell liquid culture assay. Four CD34+ cell populations, ie, (1) CD38-, HLA-DR+, (2) CD38-, HLA-DR-, (3) CD38+, HLA-DR-, and (4) CD38+, HLA-DR+ cells, were sorted as single cells into 96-well flat-bottom culture plates containing long-term culture medium supplemented with interleukin-3, interleukin-6, stem cell factor (SCF), granulocyte-macrophage colony-**stimulating** factor, erythropoietin, basic fibroblast growth factor (bFGF), and insulin-like growth factor-1 (IGF-1). Single CD34+, CD38-, HLA-DR+ cells had the highest replating efficiency. The cellular composition of the single-cell progeny was studied by morphol. and/or flow cytometric examination Only the progeny of single CD34+ cells that lacked CD38 could give rise to each of the **hematopoietic** cell lineages. The expansion of the progeny of single CD34+, CD38-, HLA-DR+ cells was examined in more detail and showed three clearly distinguishable growth patterns: 28% (SD, ±10%; n = 14) of the single cells formed cell clusters/colonies; 9% (SD, ±4%; n = 14) formed dispersed cells; and 11% (SD, ±6%; n = 14) gave rise to a mixture of cell clusters and dispersed cells. The dispersed cell growth pattern was reduced when SCF or bFGF and IGF-1 was absent in the growth factor cocktail. The replating ability of the dispersed cells was considerably larger than that of cells with other growth patterns, in that 76% of the cells that gave rise to dispersed cells and 54% of the cells that gave rise to dispersed cells as well as cell clusters gave rise to a second generation but only 7% of the cells that gave rise to cell clusters gave rise to the second generation. The second generation of cells continued to produce third and fourth generations after repetitive replating, except for the replated cells from cell clusters. In contrast with the first-generation progeny, SCF did not have an influence on the replating ability of the cells. Only in the progeny of single CD34+, CD38-, HLA-DR+ cells that gave rise to dispersed cells was each of the **hematopoietic** cell lineages found, i.e., B lymphocytes, neutrophils, monocytes, macrophages, osteoclasts, **basophils**, **mast** cells, eosinophils, erythrocytes, **megakaryocytes**, and platelets. These data suggest that the **hematopoietic** stem cells in fetal bone marrow reside within the CD34+, CD38-, HLA-DR+ cell population, but only 9% of these cells showed myeloid as well as lymphoid potential using these culture conditions. Further purification of this already miniscule cell population may therefore still be possible.
ST **hematopoietic** stem cell cytokine
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(CD34+ HLA-DR+ CD38- **hematopoietic** stem cell differentiation response to, of humans)
IT **Hematopoietic** precursor cell
(CD34+ HLA-DR+ CD38- subset, cytokines effect on, of humans)
IT **Hematopoiesis**
(cytokines effect on, by CD34+ HLA-DR+ CD38- **hematopoietic** stem cells, of humans)

IT Hemopoietins
RL: BIOL (Biological study)
(**hematopoietic** cell growth factors KL, CD34+ HLA-DR+ CD38- **hematopoietic** stem cell differentiation response to, of humans)

IT 67763-96-6, Insulin-like growth factor I 106096-93-9, Basic fibroblast growth factor
RL: BIOL (Biological study)
(CD34+ HLA-DR+ CD38- **hematopoietic** stem cell differentiation response to, of humans)

ANSWER 3 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1994:480453 CAPLUS
DN 121:80453
ED Entered STN: 20 Aug 1994
TI Interleukin 3 (IL-3)
AU Kusumi, Akinori; Kumagai, Katsuo
CS Sch. Dent., Tohoku Univ., Japan
SO Gan to Kagaku Ryoho (1994), 21(6), 915-25
CODEN: GTKRDX; ISSN: 0385-0684
DT Journal; General Review
LA Japanese
CC 15-0 (Immunochemistry)
AB A review with 53 refs. Interleukin 3 (IL-3) was initially described in the supernates of cultures of viral-infected murine spleen cells, as a cytokine produced by T lymphocytes can promote differentiation of immature T lymphocytes. Later, it was found that IL-3 exhibited a striking effect on **hematopoiesis**. The recombinant mol. of murine and human IL-3 can promote the sustained proliferation of clones of **mast** cells and **basophils**. It also acts as a colony **stimulating** factor (CSF) for bone marrow cells. Although other CSFs generally **stimulate** specific lineages of myeloid or erythroid cells, IL-3 **stimulates** bone marrow to induce proliferation of a variety of clonal cell populations, including colonies of granulocytes, macrophages, **megakaryocytes**, eosinophils, **basophils**, **mast** cells, normoblasts and erythroblasts. Thus, IL-3 is responsible for promoting proliferation of earlier lineage pluripotent stem cells, of **hematopoietic** cells and lymphoid cells. Recently, it is also suggested, as to its effects on lymphocytes, that IL-3 may possibly be a factor responsible for T lymphocytes to be differentiating extrathymically.
ST interleukin 3 **hematopoiesis** review
IT **Hematopoiesis**
(interleukin-3 effect on)
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(interleukin 3, **hematopoiesis** in response to)

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ST interleukin 3 **hematopoiesis** review
IT **Hematopoiesis**
(interleukin-3 effect on)
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(interleukin 3, **hematopoiesis** in response to)

ANSWER 2 OF 2 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

AN 92012974 EMBASE
DN 1992012974
TI Interleukin-3: Its biology and potential uses in pediatric **hematology**/oncology.
AU Sunderland M.C.; Roodman G.D.
CS Hematology Research, VA Medical Center, 7400 Merton Minton Blvd., San Antonio, TX 78284, United States
SO American Journal of Pediatric Hematology/Oncology, (1991) 13/4 (414-425). ISSN: 0192-8562 CODEN: APHODH
CY United States
DT Journal; Conference Article
FS 025 Hematology
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB The **hematopoietic** growth factor interleukin (IL)-3 is a potent regulator of blood cell proliferation. It promotes the survival, proliferation, and development of **hematopoietic** stem cells and committed progenitor cells of the granulocyte-macrophage, erythrocyte, eosinophil, **basophil, megakaryocyte, mast** cell, and lymphocyte lineages. In addition, IL-3 enhances mature myeloid cell functions such as phagocytosis and **activation** of **basophils** and eosinophils, as well as monocyte cytotoxicity. The first phase of clinical trials suggested that IL-3 may augment myelopoiesis in a number of clinical conditions. It may be efficacious for treatment of primary marrow disorders, including myelodysplastic syndromes and aplastic anemia. However, replacement therapy with IL-3 alone is probably not sufficient to obtain maximal stimulation of myelopoiesis. Preclinical and clinical studies published to date suggest that sequential use or combinations of growth factors will be needed to obtain optimal **hematopoietic** responses.
CT Medical Descriptors:
***hematopoiesis**
*myelopoiesis
aplastic anemia: DT, drug therapy
bone marrow disease
conference paper
drug activity
influenza: SI, side effect
molecular biology
myelodysplasia: DT, drug therapy
Drug Descriptors:
*interleukin 3: PD, pharmacology
*interleukin 3: DT, drug therapy
*interleukin 3: DV, drug development
*interleukin 3: EC, endogenous compound
*interleukin 3: AE, adverse drug reaction
*interleukin 3: DO, drug dose

ANSWER 2 OF 2 EMBASE
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(cytokines effect on, by CD34+ HLA-DR+ CD38- **hematopoietic** stem cells, of humans)

IT Hemopoietins
RL: BIOL (Biological study)
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IT 67763-96-6, Insulin-like growth factor I 106096-93-9, Basic fibroblast growth factor
RL: BIOL (Biological study)
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